CENTERS FOR DISEASE CONTROL

MNNR

MORBIDITY AND MORTALITY WEEKLY REPORT

January 22, 1988 / Vol. 37 / No. 2

- 13 ACIP Update: Prevention of
- Haemophilus influenzae Type b Disease
 17 PCB Contamination of Ceiling Tiles —
 Madison, Wisconsin
- 19 Compendium of Animal Rabies Control,
- 31 Update: Influenza Activity United States

Recommendations of the Immunization Practices Advisory Committee (ACIP)

Update: Prevention of Haemophilus influenzae Type b Disease

Haemophilus b Conjugate Vaccine (Diphtheria Toxoid-Conjugate) has recently been licensed for use in children 18 months of age or older for the prevention of *Haemophilus influenzae* type b (Haemophilus b) disease. This vaccine consists of Haemophilus b capsular polysaccharide covalently linked to diphtheria toxoid (conjugate vaccine).

A previously developed vaccine consisting of the Haemophilus b capsular polysaccharide alone (polysaccharide vaccine) was shown to be effective in Finnish children over 24 months of age (1), the age group in which approximately 20% of all invasive Haemophilus b infections among U.S. children less than 5 years of age can be expected to occur (2). A similar, but not identical, polysaccharide vaccine was licensed for use in the United States in April 1985 on the basis of data demonstrating biochemical characteristics and immunogenicity comparable to the vaccine used in the original Finnish trial (3). In that Finnish trial, polysaccharide vaccine was not effective in children less than 18 months of age. Because of the small sample size, efficacy could not be demonstrated in children 18 to 23 months of age. Polysaccharide vaccine was immunogenic (as measured by antibody production) in children 18 to 23 months old, but less so than it was in older children (1).

Conjugate vaccine was developed with the ultimate goal of providing an effective vaccine for infants and younger children. Preliminary data from a new Finnish study suggest that conjugate vaccine was 87% effective in preventing Haemophilus b disease when administered in a three-dose regimen to infants 3 to 6 months of age (4). However, licensure of conjugate vaccine for use in infants in the United States cannot be considered until this and other efficacy trials are further evaluated. Since antibody production after vaccination with conjugate vaccine in children 18 months of age or older is substantially greater than that after vaccination with polysaccharide vaccine, conjugate vaccine has been licensed for use in these children.

Safety

When conjugate vaccine alone was given to over 1,000 adults and children, no serious adverse reactions were obsolved (5-12). When conjugate vaccine was given with diphtheria and tetanus toxoid and pertussis vaccine (DTP) and inactivated polio vaccine (IPV) to 30,000 infants, the rate and extent of serious adverse reactions did not

Haemophilus influenzae - Continued

differ from those seen when DTP was administered alone (4). In one study of over 500 children 15 to 24 months of age, no significant difference in local or systemic side effects occurred between groups of children vaccinated with either polysaccharide vaccine or conjugate vaccine (7). Local reactions were noted for 10.3% of children receiving polysaccharide vaccine and 12.5% of children receiving conjugate vaccine, while moderate fever (temperature >39.0 °C [>102.2 °F]) occurred in 1.4% of children vaccinated with polysaccharide vaccine and 0.7% of children vaccinated with conjugate vaccine.

Immunogenicity

In several studies using different regimens of vaccine administration, conjugate vaccine has shown greater immunogenicity than polysaccharide vaccine (5-9.11,12). Response to a single dose of either polysaccharide vaccine or conjugate vaccine in children 15 to 24 months of age was specifically addressed in a randomized, doubleblind study recently completed in the United States (7). More than 90% of children vaccinated with conjugate vaccine responded with antibody levels considered to be protective (0.15 µg/mL), whereas less than 50% of children vaccinated with polysaccharide vaccine had such a response. Over 60% of children vaccinated with conjugate vaccine, but less than 30% of those vaccinated with polysaccharide vaccine, produced levels of antibody considered to be indicative of long-term protection (1.0 µg/mL).* Children given conjugate vaccine at 15 to 24 months of age had significantly higher levels of antibody to Haemophilus b polysaccharide 1 year after vaccination than did children receiving polysaccharide vaccine (8). Conjugate vaccine recipients responded to a booster dose of either polysaccharide vaccine or conjugate vaccine with higher geometric mean antibody levels than did those initially vaccinated with polysaccharide vaccine (8).

In another study, children with sickle cell syndromes who received conjugate vaccine had higher postvaccination levels of antibody to Haemophilus b polysaccharide than did similar children given polysaccharide vaccine (13). The studies to date showing increased immunogenicity in children less than 18 months of age (5,6,9,11) suggest that conjugate vaccine may be functioning as a T-cell dependent antigen. This finding contrasts with the lack of immunogenicity in infants and the absence of immunologic memory characteristic of T-cell independent polysaccharide vaccines.

Biological Activity

Several investigators have demonstrated that conjugate vaccine produces functional activity against Haemophilus b similar to that produced by polysaccharide vaccine. In one randomized, double-blind study, adults vaccinated with conjugate vaccine had serum bactericidal titers for Haemophilus b at least as high as those of adults receiving polysaccharide vaccine (12). In addition, sera from adults vaccinated with conjugate vaccine were protective in an infant rat model of Haemophilus b

^{*}It should be noted that three of four lots of polysaccharide vaccine used in this study had been heat-sized, a process which may reduce immunogenicity. However, children receiving non-heat-sized polysaccharide vaccine also had postimmunization levels of antibodies to Haemophilus b polysaccharide that were lower than those observed in children vaccinated with conjugate vaccine. In another study in which vaccine recipients were tested at 1 month and again at 1 year after completion of the immunization series, 9- to 15-month-old children who had received two doses of conjugate vaccine had significantly higher titers of antibody to Haemophilus b polysaccharide than did similar children who had received two doses of non-heat-sized polysaccharide vaccine (5).

Haemophilus influenzae - Continued

disease, whereas similarly diluted sera from persons receiving polysaccharide vaccine showed no protective activity. In a separate study, sera from 9- to 14-month-old children given conjugate vaccine showed greater opsonic activity against Haemophilus b organisms than did sera from children vaccinated with polysaccharide vaccine (14). Both studies showed a correlation between functional activity and serum levels of antibody to Haemophilus b polysaccharide and suggest that antibody produced in response to conjugate vaccine is biologically equivalent to that produced in response to polysaccharide vaccine.

Immunization Practices Advisory Committee (ACIP) Recommendations

- 1. The ACIP recommends that all children receive conjugate vaccine at 18 months of age. The efficacy of conjugate vaccine in children 18 months of age or older has not been determined in field trials. However, studies comparing antibody production in children receiving conjugate vaccine with that in children receiving polysaccharide vaccine suggest that conjugate vaccine is likely to be more effective than polysaccharide vaccine. The ACIP therefore recommends use of conjugate vaccine in all children vaccinated against Haemophilus b disease.
- 2. While the duration of immunity after a single dose of conjugate vaccine is unknown at this time, it is expected to be at least 1.5 to 3 years. Until further information is available, revaccination is not recommended for children receiving conjugate vaccine at 18 months of age or older.
- 3. Vaccination of children more than 24 months of age who have not yet received Haemophilus b vaccine should be based on risk of disease. Children considered at high risk for Haemophilus b disease, including those attending day-care centers, those with anatomic or functional asplenia (i.e., sickle cell disease or splenectomy), and those with malignancies associated with immunosuppression, should receive the vaccine. Although risk of disease decreases with increasing age, physicians may wish to vaccinate previously healthy children between 2 and 5 years of age to prevent disease that can occur in this group.
- 4. Because many children who received polysaccharide vaccine between the ages of 18 and 23 months may have had a less than adequate response to the vaccine, they should be revaccinated with a single dose of conjugate vaccine. Revaccination should take place a minimum of 2 months after the initial dose of polysaccharide vaccine.
- There is no need to routinely revaccinate children who received polysaccharide vaccine at 24 months of age or older.
- Children who had invasive Haemophilus b disease when they were less than 24 months of age should still receive vaccine according to the above recommendations since most children less than 24 months of age fail to develop adequate immunity following natural infection (15).
- 7. Although increases in serum diphtheria anti-toxin levels can follow administration of conjugate vaccine, this vaccine should not be considered an immunizing agent against diphtheria. No changes in the schedule for administration of diphtheria toxoid, customarily given as DTP, should be made secondary to the use of conjugate vaccine.
- 8. Vaccination with either polysaccharide vaccine or conjugate vaccine probably does not inhibit asymptomatic carriage of Haemophilus b organisms. Although vaccinated children may be protected from invasive disease, they may pass the organism on to susceptible children. In addition, no vaccine is 100% effective.

Haemophilus influenzae - Continued

Therefore, chemoprophylaxis of household or day-care contacts of children with Haemophilus b disease should be directed at vaccinated as well as unvaccinated contacts. Because of the length of time necessary to generate an immunologic response to the vaccines, vaccination does not play a major role in the management of patients with Haemophilus b disease or their contacts. Vaccine may be given to previously unvaccinated children of appropriate age to provide protection against future exposure.

9. Conjugate vaccine and DTP may be given simultaneously at different sites. Data are lacking on concomitant administration of conjugate vaccine and measles-mumps-rubella (MMR) or oral polio (OPV) vaccines. However, if the recipient is unlikely to return for further vaccination, simultaneous administration of all vaccines appropriate to the recipient's age and previous vaccination status is recommended (including DTP, OPV, MMR, and conjugate vaccine).

References

- Peltola H, Käyhty H, Virtanen M, Mäkelä PH. Prevention of Hemophilus influenzae type b bacteremic infections with the capsular polysaccharide vaccine. N Engl J Med 1984;310: 1561-6.
- Cochi SL, Broome CV, Hightower AW. Immunization of U.S. children with Hemophilus influenzae type b polysaccharide vaccine: a cost-effectiveness model of strategy assessment. JAMA 1985;253:521-9.
- Immunization Practices Advisory Committee. Polysaccharide vaccine for prevention of Haemophilus influenzae type b disease. MMWR 1985;34:201-5.
- Eskola J, Peltola H, Takala AK, et al. Efficacy of Haemophilus influenzae type b polysaccharide-diphtheria toxoid conjugate vaccine in infancy. N Engl J Med 1987;317: 717-22.
- Lepow ML, Samuelson JS, Gordon LK. Safety and immunogenicity of Haemophilus influenzae type b-polysaccharide diphtheria toxoid conjugate vaccine in infants 9 to 15 months of age. J Pediatr 1985;106:185-9.
- Käyhty H, Eskola J, Peltola H, Stout MG, Samuelson JS, Gordon LK. Immunogenicity in infants of a vaccine composed of *Haemophilus influenzae* type b capsular polysaccharide mixed with DPT or conjugated to diphtheria toxoid. J Infect Dis 1987;155:100-6.
- Berkowitz CD, Ward JI, Meier K, et al. Safety and immunogenicity of Haemophilus influenzae type b polysaccharide and polysaccharide diphtheria toxoid conjugate vaccines in children 15 to 24 months of age. J Pediatr 1987;110:509-14.
- Berkowitz CD, Ward JI, Hendley JO, et al. Persistence of antibody (AB) to Haemophilus influenzae type b (Hib) and response to PRP and PRP-D booster immunization in children initially immunized with either vaccine at 15 to 24 months [Abstract no. 889]. Pediatr Res 1987:21:3214
- Eskola J, Käyhty H, Peltola H, et al. Antibody levels achieved in infants by course of Haemophilus influenzae type b polysaccharide/diphtheria toxoid conjugate vaccine. Lancet 1985:1:1184-6.
- Lepow ML, Barkin RM, Berkowitz CD, et al. Safety and immunogenicity of Haemophilus influenzae type b polysaccharide-diphtheria toxoid conjugate vaccine (PRP-D) in infants. J Infect Dis 1987;156:591-6.
- Lepow ML, Randolph M, Cimma R, et al. Persistence of antibody and response to booster dose of *Haemophilus influenzae* type b polysaccharide diphtheria toxoid conjugate vaccine in infants immunized at 9 to 15 months of age. J Pediatr 1986;108:882-6.
- Granoff DM, Boies EG, Munson RS. Immunogenicity of Haemophilus influenzae type b polysaccharide-diphtheria toxoid conjugate vaccine in adults. J Pediatr 1984;105:22-7.
- Frank AL, Labotka RJ, Frisone LR, et al. H. influenza b immunization of children with sickle cell diseases [Abstract no. 906]. Pediatr Res 1987;21:324A.
- Cates KL. Serum opsonic activity for Haemophilus influenzae type b in infants immunized with polysaccharide-protein conjugate vaccines. J Infect Dis 1985;152:1076-7.
- Immunization Practices Advisory Committee. Update: prevention of Haemophilus influenzae type b disease. MMWR 1986;35:170-4,179-80.

Epidemiologic Notes and Reports

PCB Contamination of Ceiling Tiles - Madison, Wisconsin

In November of 1986, a manufacturer of ceiling tiles notified a local public school in Madison, Wisconsin, that the school contained ceiling tiles contaminated with polychlorinated biphenyl (PCB) compounds. The manufacturer offered to replace the tiles, and the Wisconsin Department of Health and Social Services, in cooperation with Madison officials and local representatives of the manufacturer, investigated the potential health hazard. The study included environmental monitoring before and after removal of the contaminated tiles and an analysis of PCB levels in serum samples from the school's staff.

Ceiling tiles manufactured by this company were first identified as a source of PCB contamination of air and surfaces during an investigation of a fire at a community college in New Jersey (1). Following this fire, the company reported that PCB-containing tiles had been manufactured in limited quantities in 1969 and 1970. The Madison, Wisconsin, public school was one of four sites in the United States known to contain these tiles.

Before removal of the tiles, air concentrations of PCB (quantified as an isomeric mixture containing 54% chlorine by weight) in the Madison school ranged from 1.6 micrograms per cubic meter ($\mu g/M^3$) to 5.1 $\mu g/M^3$ (time-weighted average [TWA] over 17 hours) in areas where ceilings were constructed entirely from the contaminated tiles (mean = 2.7 $\mu g/M^3$). The air in areas with PCB-containing tiles only around the perimeter of the ceiling had intermediate levels of PCBs (mean = 1.4 $\mu g/M^3$, range = 1.1 $\mu g/M^3$ to 1.8 $\mu g/M^3$). The air in areas without any PCB-containing tiles had lower concentrations of PCBs (mean = 1.0 $\mu g/M^3$, range = 0.9 $\mu g/M^3$ to 1.0 $\mu g/M^3$). A total of 17 air samples were analyzed.

In December 1986, after this initial environmental testing, local officials requested that the manufacturer replace the contaminated tiles as soon as possible. The school's staff and students temporarily moved to a new location in January 1987. Then, using isolation techniques similar to those used in asbestos abatement, the manufacturer removed approximately 30,000 square feet of ceiling tile and cleaned the remaining exposed surfaces. Air monitoring following tile removal showed diminished PCB concentrations. Mean PCB levels in areas where ceilings were constructed entirely from contaminated tiles decreased to 1.1 $\mu\text{g/M}^3$. The mean concentration in areas with PCB-containing tiles only around the perimeter of the ceiling decreased to 1.3 $\mu\text{g/M}^3$. Mean concentration in areas with no PCB-containing tiles decreased to 0.7 $\mu\text{g/M}^3$. New ceiling tiles were then installed, and classes resumed February 17, 1987.

Additional monitoring in May 1987 showed further decreases in air concentrations of PCBs. Areas that had contained only contaminated ceiling tiles had an average concentration of $0.7 \mu g/M^3$; areas with contaminated tiles only around the perimeter had an average concentration of $0.8 \mu g/M^3$.

The school had been occupied for 16 years, and the average length of employment at that location for the 59 current staff members was 7 years. Serum samples from 73 current and former staff members were biologically monitored to determine whether increased PCB absorption could be detected. When the capillary column method of analysis (2) was used, the geometric mean of all PCB congeners was 1.2 micrograms per liter (µg/L) of blood (range = nondétectable to 12.2 µg/L).* In a Finnish

PCB Contamination - Continued

study using the same laboratory method, the geometric mean for PCB congeners was $1.2 \pm 0.6 \mu g/L$ for people with no specific exposure to PCBs (2).

Reported by: J Schmidt, PhD, Madison Dept of Public Health; M Rubenstein, PhD, W Sonzogni, PhD, Wisconsin State Laboratory of Hygiene; J Schirmer, MS, H Anderson, MD, Environmental Epidemiologist, Wisconsin Dept of Health and Social Svcs. Div of Field Svcs, Epidemiology Program Office; Div of Environmental Hazards and Health Effects, Center for Environmental Health and Injury Control: Office of the Director, National Institute for Occupational Safety and Health, CDC.

Editorial Note: Although epidemiologic evidence remains inconclusive (3), the International Agency for Research on Cancer has suggested that PCBs be considered "probable" human carcinogens (4), and animal studies indicate a potential for adverse reproductive effects (5-8). For airborne PCBs, the Occupational Safety and Health Administration has promulgated permissible 8-hour TWA exposure limits of 0.5 mg/M3 for PCBs containing 54% chlorine and 1 mg/M3 for PCBs containing 42% chlorine (9). The National Institute for Occupational Safety and Health (NIOSH), CDC, has recommended that occupational exposure by inhalation be limited to an 8-hour TWA ≤1.0 µg total PCBs/M3 (the minimum reliably detectable concentration using the recommended sampling and analytical methods) (10). The initial environmental sampling at the school indicated that PCB concentrations in most working areas exceeded the NIOSH recommended limit. Although PCB air concentrations were lower immediately following removal of the contaminated tiles, they remained above the NIOSH recommended exposure limit. Several months following tile removal, repeat sampling for PCBs documented air concentrations below the NIOSH recommended limit.

The levels of PCBs in serum samples from staff members were similar to levels previously reported in various populations with no known specific exposures to PCBs (11,12). Environmental data were not available to characterize past exposures in the study population, nor were biological data available to characterize the staff's PCB body burden before exposure to the ceiling tiles. PCB in the serum samples could have been related primarily to accumulation from other sources such as diet (13), with some unknown additional contribution from exposures attributable to the contaminated ceiling tiles. The PCB blood values were well below levels that have been observed in occupational groups that have an increased prevalence of abnormal liver enzymes, one of the subtle effects suggestive of chronic PCB exposure (14).

References

 Centers for Disease Control. PCB contamination of ceiling tiles in public buildings – New Jersey. MMWR 1986;36:89-91.

 Luotamo M, Järvisalo J, Aitio A. Analysis of polychlorinated biphenyls (PCBs) in human serum. Environ Health Perspect 1985;60:327-32.

 Brown DP, Jones M. Mortality and industrial hygiene study of workers exposed to polychlorinated biphenyls. Arch Environ Health 1981;36:120-9.

 International Agency for Research on Cancer. The evaluation of the carcinogenic risk of chemicals to humans: polychlorinated biphenyls and polybrominated biphenyls. Vol. 18. Geneva: World Health Organization, 1978.

 Villeneuve DC, Grant DL, Khera K, Clegg DJ, Baer H, Phillips WE. Fetotoxicity of a polychlorinated biphenyl mixture (Aroclor 1254) in the rabbit and in the rat. Environ Physiol 1971;1:67-71.

 Allen JR, Carstens LA, Barsotti DA. Residual effects of short-term, low-level exposure of nonhuman primates to polychlorinated biphenyls. Toxicol Appl Pharmacol 1974;30:440-51.

 Barsotti DA, Marlar RJ, Allen JR. Reproductive dysfunction in rhesus monkeys exposed to low levels of polychlorinated biphenyls (Aroclor 1248). Food Cosmet Toxicol 1976; 14:99-103.

PCB Contamination - Continued

- Allen JR, Barsotti DA. The effects of transplacental and mammary movement of PCBs on infant rhesus monkeys. Toxicol 1976;6:331-40.
- Centers for Disease Control. NIOSH recommendations for occupational safety and health standards. MMWR 1986:35(suppl 1S):27S.
- National Institute for Occupational Safety and Health. Criteria for a recommended standard: occupational exposure to polychlorinated biphenyls (PCBs). Cincinnati: US Department of Health, Education, and Welfare, Public Health Service, 1977; DHEW publication no. (NIOSH)77-225.
- Landrigan PJ. General population exposure to environmental concentrations of halogenated biphenyls. In: Kimbrough RD, ed. Halogenated biphenyls, terphenyls, naphthalenes, dibenzodioxins and related products. New York: Elsevier/North-Holland Biomedical Press, 1980:267-86.
- Wolff MS. Occupational exposure to polychlorinated biphenyls (PCBs). Environ Health Perspect 1985;60:133-8.
- Kreiss K, Roberts C, Humphrey HEB. Serial PBB levels, PCB levels, and clinical chemistries in Michigan's PBB cohort. Arch Environ Health 1982;37:141-7.
- Maroni M, Colombi A, Arbosti G, Cantoni S, Foa V. Occupational exposure to polychlorinated biphenyls in electrical workers. II health effects. Br J Ind Med 1981;38:55-60.

Current Trends

Compendium of Animal Rabies Control, 1988 Prepared by: The National Association of State Public Health Veterinarians, Inc.*

Part I: Recommendations for Immunization Procedures

The purpose of these recommendations is to provide information on rabies vaccines to practicing veterinarians, public health officials, and others concerned with rabies control. This document will serve as the basis for animal rabies vaccination programs throughout the United States. Its adoption should result in standardization of procedures among jurisdictions, which is necessary for an effective national rabies control program. These recommendations are reviewed and revised as necessary prior to the beginning of each calendar year. All animal rabies vaccines licensed by the U.S. Department of Agriculture and marketed in the United States are listed in Part II of the compendium. Part III describes the principles of rabies control.

A. Vaccine Administration

It is recommended that all animal rabies vaccines be restricted to use by or under the supervision of a veterinarian.

B. Vaccine Selection

In comprehensive rabies control programs, it is recommended that only vaccines with a 3-year duration of immunity be used. This practice eliminates the need for annual vaccination and constitutes the most effective method of increasing the proportion of immunized dogs and cats. (See Part II.)

*THE NASPHV COMPENDIUM COMMITTEE: R. Keith Sikes, DVM, MPH, Chairman; Russell W. Currier, DVM, MPH; Suzanne Jenkins, VMD, MPH; Russell J. Martin, DVM, MPH; Grayson B. Miller, Jr., MD; F. T. Satalowich, DVM, MSPH; James M. Shuler, DVM, MPH. CONSULTANTS TO THE COMMITTEE: Melvin K. Abelseth, DVM, PhD, New York State Department of Health; Kenneth L. Crawford, DVM, MPH; Thomas R. Eng, VMD, MPH, Centers for Disease Control; David A. Espeseth, DVM, Veterinary Biologics Staff, APHIS, U.S. Department of Agriculture; Paul Waters, Representative, Veterinary Biologics Section, Animal Health Institute. ENDORSED BY: Council of State and Territorial Epidemiologists; AVMA Council on Public Health and Regulatory Veterinary Medicine.

C. Route of Inoculation

Unless otherwise specified by the product label or package insert, all vaccines must be administered intramuscularly at one site in the thigh.

D. Wildlife Vaccination

Vaccination of wildlife is not recommended since no rabies vaccine is licensed for use in wild animals and since there is no evidence that any vaccine will protect wild animals against rabies. It is recommended that neither wild nor exotic animals be kept as pets. Offspring born to wild animals bred with domestic dogs or cats are considered wild animals.

E. Accidental Human Exposure to Vaccine

Accidental inoculation of individuals may occur during administration of animal rabies vaccine. Such exposure to inactivated vaccines constitutes no rabies hazard. No cases of rabies have resulted from needle or other exposure to a licensed modified live-virus vaccine in the United States.

(Continued on page 25)

TABLE I. Summary - cases of specified notifiable diseases, United States

	2n	d Week End	ing	Cumulat	ive, 2nd We	ek Ending
Disease	Jan. 16, 1988	Jan. 17, 1987	Median 1983-1987	Jan. 16, 1988	Jan. 17, 1987	Median 1983-1987
Acquired Immunodeficiency Syndrome (AIDS)	525	219	66	925	482	152
Aseptic meningitis Encephalitis: Primary (arthropod-borne	43	87	91	98	204	167
& unspec)	6	12	12	12	33	33
Post-infectious			-	1		2
Gonorrhea: Civilian	10,380	18,878	17,789	22,212	36,885	31,485
Military	158	239	437	310	713	713
Hepatitis: Type A	160	414	329	407	679	605
Type B	175	381	375	356	716	704
Non A, Non B	19	59	57	38	114	108
Unspecified	10	59	74	30	94	122
Legionellosis	7	13	12	11	37	21
Leprosy	3	9	4	3	9	10
Malaria	4	12	9	10	31	16
Measies: Total®	1	7	7	10	42	18
Indigenous	1	2	5 2	9	40	16
Imported	24	76	-2	70	135	2
Meningococcal infections Mumps	34 33	120	55 67	70	213	104
Pertussis	8	138 33	30	90 25	59	21 10 16 18 16 2 90 104 57
Rubella (German measies)	1	2	6	23	5	9
Syphilis (Primary & Secondary): Civilian	478	541	535	913	1,239	889
Military	3		5	4	2	6
Toxic Shock syndrome	4	6	8	6	9	14
Tuberculosis	185	329	283	279	547	496
Tularemia	4	1	1	4	3	3
Typhoid Fever	1	-	5	2	4	7
Typhus fever, tick-borne (RMSF)		-	1	-	4	3
Rabies, animal	37	57	75	70	124	124

TABLE II. Notifiable diseases of low frequency, United States

	Cum. 1988		Cum. 1988
Anthrax Botulism: Foodborne		Leptospirosis (Mass. 1)	2
Infant Other		Poliomyelitis, Paralytic Paittacosis	
Brucellosis Cholera	1	Rabies, human Tetanus	
Congenital rubella syndrome		Trichinosis	1
Congenital syphilis, ages < 1 year Diphtheria	1:		

^{*}There were no cases of internationally imported measles reported for this week.

TABLE III. Cases of specified notifiable diseases, United States, weeks ending January 16, 1988 and January 17, 1987 (2nd Week)

		Aseptic	Ences	halitis	-		Н	iapatitis	(Viral), by	type		
Reporting Area	AIDS	Menin- gitis	Primary	Post-in- fectious	Gono (Civi		A	В	NA,NB	Unspeci- fied	Legionei- losis	Leprosy
	Cum. 1988	Cum. 1988	Cum. 1988	Cum. 1988	Cum. 1988	Cum. 1987	Cuin. 1988	Cum. 1988	Cum. 1988	Cum. 1988	Cum. 1988	Cum. 1988
UNITED STATES	925	98	12	1	22,212	36,885	407	356	38	30	11	3
NEW ENGLAND	84	8		-	810	1,136	21	36	7	7		2
Maine	1	1		•	21	12		2		1	*	
N.H. Vt.	3	4			24	13	2	1	1			
Mass.	56	1			249	406	14	30	5	6		2
R.I.	4	1	*		71	112	5	3	1	*	*	
Conn.	20		*		437	585						
MID. ATLANTIC	177	17			1,723	6,463 328	34 27	35 11	4	2	2 2	1
Upstate N.Y. N.Y. City	86 87	8			1,050	4,298	21	13		2		1
N.J.	4	9			326	499	7	11	3		-	
Pa.	*	-	-	-	120	1,338			*		*	-
E.N. CENTRAL	45	28	3	-	4,115	4,190	23	70	2	4	5	
Ohio	1	11 2	2	-	1,045 466	1,154	3	20				
Ind.	43	- 2			1,238	1,284						
Mich.	-	15	1		1,301	1,130	20	50	2	4	5	
Wis.		*	*	-	65	431			*			
W.N. CENTRAL	28	1		*	1,028	1,212	40	13	1		3	
Minn.	15		*	*	184	169	1	8		*	1	
lowa Mo.	1				100 566	119 732	7	2				
N. Dak.					1	11	-	-				
S. Dak.				*	23	39	-				:	*
Nebr. Kens.	4 8	1		*	34 120	109	7 25	3	1		2	
	_		-				-					
S. ATLANTIC	146	13			5,978	10,858 126	15	62	2	1		
Md.		3			411	818		1				
D.C.	6				380	649		-				
Va. W. Va.	2	2			684	967 38	2	3 5		1		
N.C.	18	2			845	1,841	5	12				
S.C.	5	-			510	1,369	2	37	2			
Ga. Fla.	28 82	1 3	-	~	1,114	1,511 3,549	5	2				
E.S. CENTRAL	30	9	2		2,172	2,381	25 23	22	6	1	1	
Ky. Tenn.	18	2			590	792	2	12	2			
Ala.	5	3	1		847	779		7	1	1	1	*
Miss.	7	1		-	549	570	-				*	
W.S. CENTRAL	8	1	*		4,433	3,668	9	3	1			*
Ark.	7			-	262 1,777	419 443						-
La. Okla.		1		-	190	389	6	3	1			
Tex.	1				2,204	2,417	3			*	*	-
MOUNTAIN	65	5	3		449	870	114	61	7	11		
Mont.	2		*		16	17	3	3	*		*	*
ldaho Wyo.			*		11	10	8	6		-		
Colo.	1	3	1		76	219	4	1		2		
N. Mex.	4			*	76	78		8	:	-		
Ariz.	44	2	1		117	260 32	40 25	28	5 2	5		
Utah Nev.	7				131	232		6				-
PACIFIC	342	16	4	1	1,504	6,107		54	8	4		
Wash.	1	10			102	351	1	2				
Oreg.	20			:	118	211	28	12	1	1	*	*
Calif. Alaska	312	10	3	1	1,185	5,358		39	7	3		
Hawaii	7	3	1	-	38	54						-
Guam					7	8						
P.R.		1	1		61	91		16	2	1		
V.I. Amer. Samos				*	15	15			-			
		-		-	-	16				-		

TABLE III. (Cont'd.) Cases of specified notifiable diseases, United States, weeks ending January 16, 1988 and January 17, 1987 (2nd Week)

	Meleria		Meas	ies (Rut	(aloed		Manin-								
Reporting Area		Indigenous		Impo	rted*	Total	gococcal Infections	Mu	mps	1	Pertussi	18		Rubella	•
	Cum. 1988	1988	Cum. 1988	1988	Cum. 1988	Cum. 1987	Cum. 1988	1988	Cum. 1986	1988	Cum. 1988	Cum. 1987	1988	Cum. 1988	Cum 1987
UNITED STATES	10	1	9		1	42	70	33	90	8	25	59	1	2	5
NEW ENGLAND	1					1	12		2	2	2				
Maine N.H.					-		1	-	2	2	2			*	*
Vt.		-	-			1		-	-	-	-	-	-	-	
Mass.	1	-	-		*	-	8	-	*		*	-		-	
R.I. Conn.			-		*		2	-	-	-					-
MID. ATLANTIC Upetate N.Y.	2	-	-	-		1	11 5		4		*	8 7			
N.Y. City	2		-								-				
N.J.		*		*	*	1	6		4		~				
Pa.	-		*	*	-				-		-	1			
E.N. CENTRAL Ohio	1	*	*		-	19	16	13	24	~	1	12	*		1
Ind.		-		-				1	1		-	7			
101.		*						*	-	-					1
Mich. Wis.	1		*	-		19	7	12	23	-	1	1		-	*
		-	*					~	•	-	-	4	-	-	*
W.N. CENTRAL Minn.		*	*	*	-	-	1	2	4	1	4	10		~	*
lowa								-	1	1	1	2	-	-	-
Mo.		*	*		-	*	1	1	1			3		-	
N. Dek. S. Dek.	*			*			-		*	-	2	1	*	*	-
Nebr.								-	1	-	1		-	-	*
Kans.								1	1			4		-	
S. ATLANTIC					1	*	3	1	3		4	8			
Del.		*					-				1	-			
Md. D.C.			-			-	1				*	*	-	*	*
Va.			1	_			-	1	1		1	-	-		
W. Va.	*		*			*				-		1			
N.C. S.C.				*	1	*	-	*	2		2	6	*	*	
Ga.							2		-			1	-	-	-
Fla.				*		-			-	-		-	-		
E.S. CENTRAL							6	3	33	2	2	1	-		2
Ky.	*		-	*			1	1	1		100	-		-	2
Tenn. Als.	2			-			4	2	32	2	2	-	*	~	
Miss.								N	N	-		1	-	-	
W.S. CENTRAL				-	-	-	1	7	10						
Ark.		*			-						-		-	-	
Cela.	*	*	*	*			*	1	2	~		-			
Tex.				-		-	1	4 2	6 2	_	-		-	-	*
MOUNTAIN	1	1	5				3	4	4	1	2	3	1		1
Mont. Idaho			*					-	-		-				
Wyo.				:	-					*	-	2		*	*
Colo.		1	5				3	2	2			-		-	-
N. Max.	*		*	*		-		N	N	-	+	1			*
Ariz. Utah		-	*	*	*			1	1	1	1		:	:	-
Nev.	1	-				-	2	,			1		1	1	1
PACIFIC	5		4			21	17	3	6	2	10	17			
Wash.		-	-			*		2	2		10	1			
Oreg.	:	-	:	-		1	2	N	N			5		*	1
Calif. Alaska	4	-	4	-		20	14	1	3	*	2	10	*	1	-
Hawaii		-	-							2	8	1			-
Guam															
P.R.	1	-			-				2			1			
V.I. Amer. Samos	*	*	*	-	*						-				

^{*}For messles only, imported cases includes both out-of-state and international importations.

TABLE III. (Cont'd.) Cases of specified notifiable diseases, United States, weeks ending January 16, 1988 and January 17, 1967 (2nd Week)

Reporting Area	Syphilis (Primary &	(Civilian) Secondary)	Toxic- shock Syndrome	Tuber	culosis	Tula- remia	Typhoid Faver	Typhus Fever (Tick-borne) (RMSF)	Rabies
	Cum. 1988	Cum. 1987	Cum. 1988	Cum. 1988	Cum. 1987	Cum. 1988	Cum. 1988	Cum. 1988	Cum. 1988
UNITED STATES	913	1,239	6	279	547	4	2		
NEW ENGLAND	32	17	2	3	10	-	1		70
Maine N.H.	1		1		-			:	
Vt.	1		1	*	:				
Mass.	16	13		i	1 2	_	1	*	-
R.I. Conn.	14	:	-	1					-
MID. ATLANTIC		4		1	7			-	
Upstate N.Y.	194	119		92 11	115 39	*	-		10
N.Y. City	165	71		41	59			-	
N.J. Pa.	22	17		20	9				:
	1	28		20	8	-			10
E.N. CENTRAL Ohio	16	27		57	82	-	*		2
Ind.	7	1	:	18	15	*	-		
H).	5	20		15	49	2	-		
Mich. Wis.	4	:		20	15				
	-	5		4	2		-		1
W.N. CENTRAL Minn.	3	7	3	12	10	3			9
lowa		4	1	4 2	4	*	*		
Mo.		3	i	1	5	2			5
N. Dak. S. Dak.					1	-			2
Nebr.	2		1	5			-		-
Kans.	-	-			-	1	-		1
S. ATLANTIC	394	491		52	85				•
Del.	1	3			80		-	*	24
Md. D.C.	10	22		4	6				10
Va.	17	12		1 4	6		-		-
W. Va.				4	7				6
N.C. S.C.	19 15	34		-	14		-		2
Ga.	68	30 73		16	19	*	-		
Fla.	260	313	-	23	29			*	6
E.S. CENTRAL	45	56	1	35	65	1			
ζy.		-	i	19	00	1		*	1
Tenn. Ala.	28	30		**					
Mins.	17	26		16	31				1
W.S. CENTRAL	136	175				*	-	-	-
Ark.	130	8		4	7	*		*	15
La. Okia.	11	26				-	-	2	7
Tex.	8 117	133		4	-	*			1
MOUNTAIN	3			*	7	*	*		7
Mont.	3	11		3	3	*	-		9
Idaho		2		-	-				7
Wyo. Colo.	3	2		-	-			1	1
N. Mex.	3	5		1	1			-	
Ariz.		5			1				:
Utah Nev.		-	*				-		
		-		2	1		-		
PACIFIC Wash.	90	336		21	170		1		
Oreg.	4	6	-	8 7	3			-	*
Calif.	84	325		-	143	-	1	-	-
Alaska ławaii	2	1		2	7				
Guam	4			4	13	•	*		*
P.R.	25	18	-	-	2	*	*		
/.l.	1	2		5	3		*	-	2
Amer. Samos		-	-		2				-
C.N.M.I.									

TABLE IV. Deaths in 121 U.S. cities,* week ending January 16, 1988 (2nd Week)

		All Causes, By Age (Years)						1	All Cau	ses, B	y Age	Years)		P&I**	
Reporting Area	All Ages	>65	45-64	25-44	1-24	<1	Total	Reporting Area	All Ages	>65	45-84	25-44	1-24	<1	Tota
NEW ENGLAND	723	539	123	36	15	10	61	S. ATLANTIC	1,476	907	345	133	56	35	7
loston, Mass.	174	120	37	9	5	3	24	Atlanta, Ga.	299	184	73	31	8	3	1
bridgeport, Conn.	61	48	9	4			3	Baltimore, Md.	244	159	52	19	8	6	1
ambridge, Mass.	18	11	6	1	-	-	2	Charlotte, N.C.	158	95	48	8	4	3	
all River, Mass.	36 102	33 75	12	1 8	4	3	2	Jacksonville, Fla.	138	80	33	15	9	1	
lartford, Conn. owell, Mass.	27	22	3	2	-		1	Miami, Fla.	80	32	23	18	3	4	
ynn, Mass.	18	14	4	-		-		Norfolk, Va. Richmond, Va.	93	44 54	18 23	13	2 2	6	
Vew Bedford, Mass.	23	22	1	-	-	-	2	Savannah, Ga.	56	37	13	1	1	4	
New Haven, Conn.	46	30	8	6	1	1	5	St. Petersburg, Fia.	104	84	11	3	3	3	
Providence, R.I.	44	35	6	1	2		2	Tampa, Fla.	65	43	12	3	6	1	
Somerville, Mass.	12	9	3				1	Washington, D.C.	134	76	34	14	7	3	
Springfield, Mass.	41	25	10	2	2	2	2	Wilmington, Del.	31	19	5	4	3		
Waterbury, Conn. Worcester, Mass.	39 82	34 61	17	2	1	1	6	E.S. CENTRAL	873	606	167	54	16	30	4
		-		-			8	Birmingham, Ala.	125	82	25	10	4	4	
MID. ATLANTIC	3,292	2,193		303	68	75	152	Chattanooga, Tenn.	38	29	7	1		1	
Albany, N.Y.	66	47	13	3	-	3	3	Knoxville, Tenn.	77	56	17	3		1	
Allentown, Pa.	26 150	113		3		2	1 8	Louisville, Ky.	144	105	25	6	3	5	
Buffalo, N.Y. Camden, N.J.	44	30		8	1	2		Memphis, Tenn.	198	129	47	11	3	8	
Elizabeth, N.J.	17	11	5	-		î	1	Mobile, Ala.	99 33	73 25	12	9	1	4	
Erie, Pa.1	52	45		1	1	1	4	Montgomery, Ala. Nashville, Tenn.	159	107	28	14	4	6	
Jersey City, N.J.	48	30		4		1	2			180					
N.Y. City, N.Y.	1,778	1,147	363	204	37	27	74	W.S. CENTRAL	1,621	1,036	334	149	54	48	1
Hewark, N.J.	128	69		21	8	6	3	Austin, Tex.	66	43	11	6	5	1	
Paterson, N.J.	40	27		5	1		3	Baton Rouge, La. Corpus Christi, Tex.	45 77	31 57	10 18	3	*	1	
Philadelphia, Pa.	384	248		23	13	19	18	Dallas, Tex.	271	149	59	36	14	13	
Pittsburgh, Pa.1	88 41	57	24	4	2	1	2	El Paso, Tex.	89	60	15	10	1	3	
Reading, Pa. Rochester, N.Y.	126	31 94		2 4		6	11	Fort Worth, Tex	129	85	20	16	6	2	
Schenectady, N.Y.	26	21	5	-	-		1	Houston, Tex.§	308	176	74	34	13	11	
Scranton, Pa.†	32	21	8	1	1	1		Little Rock, Ark.	107	77	16	7	4	3	
Syracuse, N.Y.	122	87	22	8	1	4	7	New Orleans, La.	93	60	20	8	5		
Trenton, N.J.	58	43		- 4	2	-	2	San Antonio, Tex.	240	168	43	16	5	8	1
Utica, N.Y.	33	24		3			2	Shreveport, La.	37	26	7	.1	-	3	
Yonkers, N.Y.	35	26	-	1	1	*	6	Tulsa, Okla. MOUNTAIN	159 833	104	141	11	1	2	
E.N. CENTRAL Akron, Ohio	2,571	1,757		152	56	78	122	Albuquerque, N. Me		64	16	57	20	26	
Canton, Ohio	41	30		- 4	1	1	4	Colo. Springs, Colo.	51	33	9	3	3	3	
Chicago, III.§	564	362		45	10	22	16		192	142	35	6	1	8	
Cincinnati, Ohio	195	136		9	4	3	17	Las Vegas, Nev.	100	67	16	14	-	3	
Cleveland, Ohio	162	104		6	7	10		Ogden, Utah	29	19	7	2	1	-	
Columbus, Ohio	85	47		9	4	4		Phoenix, Ariz.	137 56	100		10	2	2	
Dayton, Ohio	147	107		8	1	2	6		54	41	11	5	2	5	
Detroit, Mich.	319	196		29	15	7	6		122	89	17	9	5	2	
Evansville, Ind. Fort Wayne, Ind.	63	48 51		4	2		10					-			
Gary, Ind.	27	18		2		1	1	PACIFIC Berkeley, Calif.	2,197	1,503	411	171	61	43	1
Grand Rapids, Mich.		42		2	3	3	7	Fresno, Calif.	29 90	21 58		2		2 2	
Indianapolis, Ind.	203	130			4	8	6		33	24		1		-	
Madison, Wis.	58	41		4	1	1	8	Honolulu, Hawaii	74	56		4	3	2	
Milwaukee, Wis.	172	128			1	5	13	Long Beach, Calif.	148	102		11	4	3	
Peoria, III.	52	42		2		1	6	Los Angeles Calif.	527	354		51	17	7	
Rockford, III.	50	37				-	3		70	37		6	4	1	
South Bend, Ind.	72	50			1	1	2		38	27		2			
Toledo, Ohio Youngstown, Ohioś	101	83			1	3	11		128	95		7	5	2	
				_				Ouchamento, cam.	181	124		16		5	
W.N. CENTRAL	818	584			18	21	53		198 186	139		18		6	
Des Moines, Iowa	70	55			3	1	3	Con Inna Calle	212	150				2	
Duluth, Minn.	29	23			1		1	Carrette things	186	128		16		3	
Kansas City, Kans. Kansas City, Mo.	127	2	1 11 8 22		2	1	1	Cartage Miles	47	37		10		2	
Lincoln, Nebr.	45	3			2		8		50	40		2	2		
Minnespolis, Minn.	86	5			3	2	8		14,404		-	_		366	
Omaha, Nebr.	116	71			5	3	3		14,404	9,714	4,600	1,097	364	366	
St. Louis, Mo.	149	10				5	11								
St. Paul, Minn.	73	6	1 8			4	8	5 [
Wichita, Kans.	86	61	0 14	3	4	5	7								

^{*}Mortality data in this table are voluntarily reported from 121 cities in the United states, most of which have populations of 100,000 or more. A death is reported by the place of its occurrence and by the week that the death certificate was filed. Fetal deaths are not included:

**Pneumonia and influenza.

**Pneumonia and influenza.

**Because of changes in reporting methods in these 3 Pennsylvania cities, these numbers are partial counts for the current week.

**Complete counts will be available in 4 to 6 weeks.

**Total includes unknown ages.

**Solata not available. Figures are estimates based on average of past 4 weeks.

F. Identification of Vaccinated Dogs

It is recommended that all agencies and veterinarians adopt the standard tag system. This practice will aid the administration of local, state, national, and international procedures. Dog license tags should not conflict in shape and color with rabies tags. It is recommended that anodized aluminum rabies tags be no less than 0.064 inches in thickness.

1. Rabies Tags.

Calendar Year	Color	Shape
1988	Red	Heart
1989	Blue	Rosette
1990	Orange	Fireplug
1991	Green	Bell

 Rabies Certificate. All agencies and veterinarians should use the National Association of State Public Health Veterinarians (NASPHV) form #50, "Rabies Vaccination Certificate," which can be obtained from vaccine manufacturers.

Part II: Vaccines Marketed in the United States and NASPHV Recommendations

Product Name	Produced By	Marketed By	For Use In*	Dosage [†]	Age at Primary Vaccination ⁶	Booster Recommended
A. MODIFIEI	LIVE VIRUS					
ENDURALL-R	NORDEN License No. 189	Norden	Dogs Cats	1 mL 1 mL	3 mos. & 1 yr. later 3 mos.	Triennially Annually
NEUROGEN- TC	BOEHRINGER INGELHEIM License No. 124	Bio-Ceutic	Dogs	1 mL	3 mos. & 1 yr. later	Triennially
B. INACTIVA	TED					
					3 mos. &	
TRIMUNE	FORT DODGE License No. 112	Ft. Dodge	Dogs	1 mL	1 yr. later 3 mos. &	Triennially
			Cats	1 mL	1 yr. later	Triennially
ANNUMUNE	FORT DODGE License No. 112	Ex Dadas	Dogs Cats	1 mL 1 mL	3 mos.	Annually
	License No. 112	Ft. Dodge	Cats	1 mL	3 mos.	Annually
BIORAB-1	SCHERING License No. 165-A	Biologics Corp.	Dogs Cats	1 mL 1 mL	3 mos.	Annually Annually
					3 mos. &	
BIORAB-3	SCHERING License No. 165-A	Biologics Corp.	Dogs Cats	1 mL 1 mL	1 yr. later 3 mos.	Triennially Annually
			_		3 mos. &	
RABMUNE 3	SCHERING License No. 165-A	Beecham	Dogs Cats	1 mL 1 mL	1 yr. later 3 mos.	Triennially Annually

^{*}Refers only to domestic species of this class of animals.

[†]All vaccines must be administered *intramuscularly* at one site in the thigh unless otherwise specified by the label.

⁵Three months of age or older and revaccinated 1 year later.

Part II: Vaccines Marketed in the United States and NASPHV Recommendations - Continued

Product Name	Produced By	Marketed By	For Use In*	Dosage [†]	Age at Primary Vaccination ⁸	Booster Recommended
DURA-RAB 1	IMMUNOVET	ImmunoVet &	Dogs	1 mL	3 mos.	Annually
	License No. 302-A	Vedco, Inc.	Cats	1 mL	3 mos.	Annually
					3 mos. &	
DURA-RAB 3	IMMUNOVET	ImmunoVet &	Dogs	1 mL	1 yr. later	Triennially
	License No. 302-A	Vedco, Inc.			3 mos. &	
			Cats	1 mL	1 yr. later	Triennially
					3 mos. &	
RABCINE 3	IMMUNOVET	-	Dogs	1 mL	1 yr. later	Triennially
	License No. 302-A	Beecham	0-1-		3 mos. &	T
			Cats	1 mL	1 yr. later	Triennially
RABCINE	BEECHAM		Dogs	1 mL	3 mos.	Annually
	License No. 225	Beecham	Cats	1 mL	3 mos.	Annually
ENDURALL-K	NORDEN		Dogs	1 mL	3 mos.	Annually
	License No. 189	Norden	Cats	1 mL	3 mos.	Annually
					3 mos. &	
RABGUARD-	NORDEN		Dogs	1 mL	1 yr. later	Triennially
TC	License No. 189	Norden			3 mos. &	
			Cats	1 mL	1 yr. later	Triennially
			Sheep	1 mL	3 mos.	Annually
			Cattle	1 mL	3 mos.	Annually
			Horses	1 mL	3 mos.	Annually
CYTORAB	COOPERS ANIMAL HEALTH, INC.		Dogs	1 mL	3 mos.	Annually
	License No. 107	Coopers	Cats	1 mL	3 mos.	Annually
TRIRAB	COOPERS ANIMAL				3 mos. &	
	HEALTH, INC.		Dogs	1 mL	1 yr. later	Triennially
	License No. 107	Coopers	Cats	1 mL	3 mos.	Annually
RABVAC 1	FROMM	Solvey	Dogs	1 mL	3 mos.	Annually
	License No. 195-A	Veterinary	Cats	1 mL	3 mos.	Annually
					3 mos. &	
RABVAC 3	FROMM	Solvay	Dogs	1 mL	1 yr. later	Triennially
	License No. 195-A	Veterinary			3 mos. &	
			Cats	1 mL	1 yr. later	Triennially
IMRAB	MERIEUX		Dogs	1 mL	3 mos.	Triennially
	License No. 298	Pitman-Moore	Cats	1 mL	& 1 yr.	Triennially
			Sheep	1 ml.	later	Triennially
			Cattle	2 mL	3 mos.	Annually
			Horses	2 mL	3 mos.	Annually
IMPAB-1	MERIEUX		Dogs	1 mL	3 mos.	Annually
	License No. 298	Pitman-Moore	Cats	1 mL	3 mos.	Annually
C. COMBINA	ATION					
ECLIPSE 3	FROMM	Solvay				
KP-R	License No. 195-A	Veterinary	Cats	1 mL	3 mos.	Annually

*Refers only to domestic species of this class of animals.

*All vaccines must be administered intramuscularly at one site in the thigh unless otherwise specified by the label.

*Three months of age or older and revaccinated 1 year later.

Part II: Vaccines Marketed in the United States and NASPHV Recommendations — Continued

Product Name	Produced By	Marketed By	For Use In*	Dosage [†]	Age at Primary Vaccination ⁸	Booster Recommended
ECLIPSE 4 KP-R	FROMM Liense No. 195-A	Solvay Veterinary	Cats	1 mL	3 mos.	Annually
CYTORAB RCP	COOPERS ANIMAL HEALTH, INC. License No. 107	Coopers	Cats	1 mL	3 mos.	Annually
FEL-O-VAX PCT-R	FORT DODGE License No. 112	Ft. Dodge	Cats	1 mL	3 mos. & 1 yr. later	Triennially
ECLIPSE 4-R	FROMM License No. 195-A	Solvay Veterinary	Cats	1 mL	3 mos.	Annually

*Refers only to domestic species of this class of animals.

[†]All vaccines must be administered intramuscularly at one site in the thigh unless otherwise specified by the label.

⁵Three months of age or older and revaccinated 1 year later.

Part III: Principles of Rabies Control

These guidelines have been prepared by the NASPHV for use by government officials, practicing veterinarians, and others who may become involved in certain aspects of rabies control. It is intended that the NASPHV annually review and revise these recommendations as necessary. Standardized control procedures are needed to deal effectively with the public health aspects of rabies.

A. Principles of Rabies Control

- 1. Human Rabies Prevention. Rabies in humans can be prevented either by eliminating exposures to rabid animals or by providing exposed persons with prompt local treatment of wounds combined with appropriate passive and active immunization. The rationale for recommending preexposure and postexposure rabies prophylaxis and details of their administration can be found in the current recommendations of the Immunization Practices Advisory Committee (ACIP), of the Public Health Service (1,2). These recommendations, along with information concerning the current local and regional status of animal rabies and the availability of human rabies biologics, are available from state health departments.
- 2. Domestic Animals. Local governments should initiate and maintain effective programs to remove strays and unwanted animals and to ensure vaccination of all dogs and cats. Since more cases of rabies are now reported annually among cats than among dogs, immunization of cats should be required. Such procedures in the United States have reduced laboratory-confirmed rabies cases in dogs from 6,949 in 1947 to 94 in 1986. The recommended vaccination procedures and the licensed animal vaccines are specified in Parts I and II of the NASPHV's annual compendium.
- 3. Rabies in Wildlife. The control of rabies among foxes, skunks, raccoons, and other terrestrial animals is very difficult. Selective reduction of these populations when indicated may be useful, but the usefulness of this procedure depends heavily upon the circumstances surrounding each rabies outbreak. (See C. Control Methods in Wild Animals.)

B. Control Methods in Domestic and Confined Animals

- Preexposure Vaccination and Management. Animal rabies vaccines should be administered only by or under the direct supervision of a veterinarian. Such administration is the only way to assure the public that the animal has been properly immunized. Within 1 month after vaccination, a peak rabies antibody titer is reached, and the animal can be considered immunized. (See Parts I and II for recommended vaccines and procedures.)
 - a. Dogs and Cats. All dogs and cats should be vaccinated against rabies beginning at 3 months of age and should be revaccinated in accordance with Part II of this compendium.
 - b. Livestock. It is not economically feasible, nor is it justified from a public health standpoint, to vaccinate all livestock against rabies. Owners of valuable animals and veterinary clinicians may consider immunizing certain livestock located in areas where wildlife rabies is epizootic or where colonies of bats exist.

c. Other Animals.

- (1) Animals Maintained in Exhibits and in Zoological Parks. Captive animals not completely excluded from all contact with local vectors of rabies can become infected with rabies. Moreover, such animals may be incubating rabies when captured. Exhibit animals, especially those carnivores and omnivores having contact with the viewing public, should be quarantined for a minimum of 180 days. Since no rabies vaccine is licensed for use in wild animals, vaccination even with inactivated vaccine is not recommended. Preexposure rabies vaccination of animal workers at such facilities is recommended. This practice may reduce the need for euthanasia of valuable animals for rabies testing after they have bitten a handler.
- (2) Wild Animals. Because of the existing risk of rabies in wild animals (especially raccoons, skunks, and foxes), the American Veterinary Medical Association, the NASPHV, and the Conference of State and Territorial Epidemiologists (CSTE) strongly recommend the enactment of state laws prohibiting the importation, distribution, and relocation of wild animals and wild animals crossbred with domestic dogs and cats. These same organizations continue to recommend the enactment of laws prohibiting the distribution or keeping of wild animals as pets. Moreover, the NASPHV and CSTE recommend that ferrets not be kept as pets since they have severely bitten many people and especially since their bites have mutilated infants. Ferrets are susceptible to rabies and could transmit it. Furthermore, the period of rabies virus shedding in infected ferrets is unknown.
- 2. Control of Stray Animals. Stray dogs or cats should be removed from the community, especially in areas where rabies is epizootic. Local health department and animal control officials can enforce the pickup of strays more efficiently if owned animals are confined or kept on leash. Strays should be impounded for at least 3 days to give owners sufficient time to reclaim animals and to determine if human exposure has occurred.

3. Quarantine.

- a. International. Present Public Health Service regulations (42 CFR No. 71.51) governing the importation of domestic felines and canines are minimal for preventing the introduction of rabid animals into the United States. All dogs and cats imported from countries with endemic rabies should be vaccinated against rabies at least 30 days before entry into the United States.[†] CDC is responsible for animals imported into the United States, and their requirements should be coordinated with interstate shipment requirements. The health authority of the state of destination should be notified of any animal conditionally admitted into its jurisdiction within 72 hours. The conditional admission into the United States of such animals must be subject to state and local laws governing rabies. Failure to comply with these requirements should be promptly reported to the director of CDC.
- b. Interstate. Prior to interstate shipment, dogs and cats should be vaccinated against rabies according to the compendium's recommendations and, preferably, should be vaccinated at least 30 days prior to shipment. While in shipment, they should be accompanied by a currently valid NASPHV Form #50, "Rabies Vaccination Certificate." One copy of the certificate should be mailed to the appropriate Public Health Veterinarian or State Veterinarian of the state of destination.
- c. Health Certificates. If a certificate is required for dogs and cats in transit, it must not replace the NASPHV rabies vaccination certificate.
- Adjunct Procedures. Methods or procedures that enhance rabies control include:
 - a. Licensure. Registration or licensure of all dogs and cats controls the number of stray animals and may, thus, be used as a means of rabies control. Frequently a fee is charged for such licensure, and revenues collected are used to maintain a rabies or animal control program. Vaccination is usually recommended as a prerequisite to licensure.
 - Canvassing of Area. Canvassing includes house-to-house calls by members
 of the animal control program to enforce vaccination and licensure
 requirements.
 - c. Citations. Citations are legal summonses issued to owners for violations, including the failure to vaccinate or license their animals.
 - d. Leash Laws. All communities should adopt leash laws that can be incorporated into their animal control ordinances.
- Postexposure Management. ANY DOMESTIC ANIMAL THAT IS BITTEN OR SCRATCHED BY A BAT OR BY A WILD, CARNIVOROUS MAMMAL THAT IS NOT AVAILABLE FOR TESTING SHOULD BE REGARDED AS HAVING BEEN EX-POSED TO A RABID ANIMAL.
 - a. Dogs and Cats. When bitten by a rabid animal, unvaccinated dogs and cats should be destroyed immediately. If the owner is unwilling to have this done, the unvaccinated animal should be placed in strict isolation for 6 months and vaccinated 1 month before being released. Dogs and cats that are currently

[†]In regard to cats, these recommendations do not conform to the official recommendations of CDC and the Public Health Service. Although domestic feline rabies has increased, there has been no evidence of increased risk of imported rabies in cats. U.S. Foreign Quarantine regulations do not require rabies vaccinations for imported cats.

vaccinated should be revaccinated immediately and observed by the owner for 90 days.

b. Livestock. All species of livestock are susceptible to rabies infection; cattle appear to be among the most susceptible of all domestic animal species. Livestock known to have been bitten by rabid animals should be destroyed (slaughtered) immediately. If the owner is unwilling to have this done, the animal should be kept under very close observation for 6 months.

Following are recommendations for owners of livestock exposed to rabid animals:

- (1) If the animal is slaughtered within 7 days of being bitten, its tissues may be eaten without risk of infection, provided liberal portions of the exposed area are discarded. Federal meat inspectors will reject for slaughter any animal that has been exposed to rabies within 8 months.
- (2) No tissues or secretions from a clinically rabid animal should be used for human or animal consumption. However, since pasteurization temperatures will inactivate rabies virus, the drinking of pasteurized milk or eating of completely cooked meat does not constitute a rabies exposure.
- 6. Management of Animals That Bite Humans. A healthy dog or cat that bites a person should be confined and observed for 10 days and evaluated by a veterinarian at the first sign of illness during confinement or before release. Any illness in the animal should be reported immediately to the local health department. If signs suggestive of rabies develop, the animal should be humanely killed, and its head should be removed and shipped, under refrigeration, for examination by a qualified laboratory designated by the local or state health department. Any stray or unwanted dog or cat that bites a person can be killed immediately; the head should be submitted, as described above, for rabies examination.

C. Control Methods in Wild Animals

Bats and wild carnivorous mammals (as well as wild animals crossbred with domestic dogs and cats) that bite people should be killed, and appropriate tissues should be sent to the laboratory for examination for rabies. A person bitten by a bat or any wild animal should immediately report the incident to a physician who can evaluate the need for antirabies treatment. (See current rabies prophylaxis recommendations of the ACIP [1,2].)

- 1. Terrestrial Mammals. Continuous and persistent government-funded programs for trapping or poisoning wildlife as a means of rabies control are not cost-effective in reducing wildlife reservoirs or rabies incidence on a statewide basis. However, limited control in high-contact areas (picnic grounds, camps, suburban areas) may be indicated for the removal of selected high-risk species of wild animals. The public should be warned not to handle wild animals. The state wildlife agency should be consulted early to manage any elimination programs in coordination with the state health department.
- 2. Bats. Rabid bats have been reported from every state except Hawaii and have caused human rabies infections in the United States. It is neither feasible nor practical, however, to control rabies in bats by areawide programs to reduce bat populations. Bats should be eliminated from houses and surrounding structures to prevent direct association with people. Such structures should then be made bat proof by sealing routes of entrance with screen or by other means.

References

- Immunization Practices Advisory Committee. Rabies prevention—United States, 1984. MMWR 1984;33:393-402,407-8.
- Immunization Practices Advisory Committee. Rabies prevention: supplementary statement on the preexposure use of human diploid cell rabies vaccine by the intradermal route. MMWR 1986;35:767-8.

Epidemiologic Notes and Reports

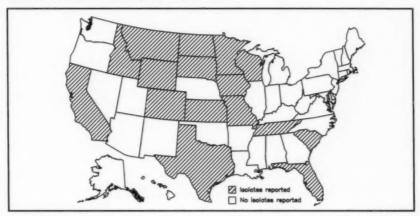
Update: Influenza Activity - United States

Influenza A(H3N2) is the most frequently reported influenza virus so far this season (Figure 1). For the report week ending January 9, 1988, seven states reported regional outbreak activity.* Widespread activity has not yet been reported this season. Sporadically occurring cases of influenza B have been reported from five states.†

Reported by: Participating State and Territorial Epidemiologists and State Laboratory Directors.

Reported by: Participating State and Territorial Epidemiologists and State Laboratory Directors. WHO Collaborating Center for Influenza, Influenza Br, Div of Viral Diseases, Center for Infectious Diseases, CDC.

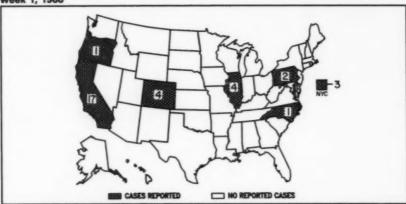
FIGURE 1. States reporting isolates of influenza A(H3N2) — United States, October 19, 1987 — January 15, 1988



^{*}Kansas, Montana, Nebraska, South Dakota, Texas, Utah, and Wisconsin.

[†]Arizona, Hawaii, New York, Ohio, and Tennessee.

FIGURE I. Reported measles cases — United States, weeks 50-52, 1987 and week 1, 1988



The Morbidity and Mortality Weekly Report is prepared by the Centers for Disease Control, Atlanta, Georgia, and available on a paid subscription basis from the Superintendent of Documents, U.S. Government Printing Office, Washington, D.C. 20402, (202) 783-3238.

The data in this report are provisional, based on weekly reports to CDC by state health departments. The reporting week concludes at close of business on Friday; compiled data on a national basis are officially released to the public on the succeeding Friday. The editor welcomes accounts of interesting cases, outbreaks, environmental hazards, or other public health problems of current interest to health officials. Such reports and any other matters pertaining to editorial or other textual considerations should be addressed to: Editor, Morbidity and Mortality Weekly Report, Centers for Disease Control, Atlanta, Georgia 30333.

Director, Centers for Disease Control James O. Mason, M.D., Dr.P.H. Director, Epidemiology Program Office Carl W. Tyler, Jr., M.D. Editor Michael B. Gregg, M.D. Managing Editor Gwendolyn A. Ingraham

☆U.S. Government Printing Office: 1988-530-111/60053 Region IV

DEPARTMENT OF HEALTH & HUMAN SERVICES Public Health Service

Public Health Service Centers for Disease Control Atlanta, GA 30333

Official Business Penalty for Private Use \$300 FIRST-CLASS MAIL POSTAGE & FEES PAID PHS/CDC Permit No. G-284

A 48106SER 06 8639 9 SERIALS ACQUISITION DEPT UNIVERSITY MICROFILMS 300 NORTH ZEEB ROAD ANN ARBOR, MI 48106

